Lack of Antireticulin and IgA Antiendomysium Antibodies in Sera of Patients with Primary IgA Nephropathy Associated with Circulating IgA Antibodies to Gliadin

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Sir,

A possible relationship of primary IgA nephropathy (IgA GN) with wheat gliadin and latent celiac disease (CD) has been suggested by the presence of high levels of circulating IgA antigliadin antibodies [5], the decrease of IgA-containing circulating immune complexes after gluten-free diet [3] and the few cases of IgA GN associated with patent CD and/or dermatitis herpetiformis (DH) [4]. Antireticulin and IgA class antiendomysium antibodies (IgA EmA) are specific immunological markers for CD and DH associated with CD [2,6]. The IgA EmA are found only in gluten-sensitive enteropathy and their formation precedes the development of the pathological gut changes in CD and DH [1]; these autoantibodies become undetectable in CD when the flattening of the gut mucosa disappears after gluten-free diet [1]. Therefore, we looked for antireticulin and IgA EmA in the sera of IgA GN patients with high levels of circulating IgA antigliadin antibodies. We analyzed the sera of 19 untreated patients with IgA GN having permanent proteinuria, according to the usual histological and immunofluorescence criteria [4], ($> 1 < 3$ g/24 h; age: mean 41 years, range 24–58). All the patients exhibited high levels of circulating IgA antigliadin antibodies (ELISA technique DO > 0.65 ; mean DO: 1.11, range 0.66–2.6 [5]); they were free of abdominal pain, diarrhea and malabsorption symptoms. All the patients were on a normal diet. The antireticulin and antiendomysium antibodies were studied with indirect immunofluorescence technique with sera tested at the dilutions of one fifth and one tenth; for antireticulin antibodies the substrates were unfixed frozen sections of rat liver, kidney and gastric mucosa [6] and for antiendomysium antibodies the substrates were frozen sections of monkey esophagus (Zeus-Scientific Inc.) [2]. The conjugates were fluorescein-conjugated antibodies to human immunoglobulins (IgG, IgA, IgM: Diagnostic Pasteur, dilution 1/100) and to human α -chain (Dako, dilution 1/20). No patients exhibited either the specific RI pattern for antireticulin antibodies or IgA antiendomysium. Therefore, the lack of antireticulin and IgA EmA specific to gluten-sensitive enteropathy in 19 patients with IgA GN and IgA antigliadin antibodies would tend to

demonstrate that no association exists between IgA GN and latent celiac disease. These results are strengthened by the normality of the gut absorption test with radiolabelled EDTA performed on 5 patients, and of the jejunal biopsies performed on 3 patients. Other investigations are necessary to explore the role of the gliadin antigens and of the IgA antigliadin antibodies in IgA nephropathy.

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